



Physical activity, sport and hypertension

Fizikai aktivitás, sport és hipertónia

István Préda

Semmelweis University, Faculty of General Medicine & M.D. Military Hospital

Absztrakt: Rendszeres fizikai terhelés hatására növekszik a koszorúér és szívizom áramlás, fokozódik a pulzustérfogat és a perctérfogat. A kedvező keringési hatás heti 150-200 perc, átlagosan 6-8 metabolikus ekvivalens (MET) összességében 3-4 alkalommal megvalósuló terhelésével érhető el. Szív és keringési betegségekben a terheléses program a rehabilitáció alapköve. Csúcsteljesítményt nyújtó versenysport és újabban rekreációs aktivitási formák (> heti 20 óra és 15-20 MET teljesítmény) olyan elektromos és funkcionális szív eltéréseket okozhatnak, amelyek hasonlóak a jobb kamrai aritmogén cardiomiopatia (ARCV) okozta elváltozásokhoz és hirtelen szívhalált indukálhatnak. Emocionális stressz és D-Típusú személyiségzavar szintén szimpatikus idegrendszeri túlsúlyt okoz. Elhúzódó extrém sportterhelés (Marathon futás, triatlon) jellegzetessége lehet a szimpatikus-paraszimpatikus patológiás interakció, mely fatális ritmuszavart, hirtelen szívhalált okozhat.

Kulcsszavak: Fizikai terhelés, versenysport, Marathon futás és triatlon, szimpatikus – paraszimpatikus interakció, hirtelen szívhalál

Abstract: Regular physical exercise increases coronary flow, augments myocardial tissue perfusion, pulse volume and cardiac output. The suggested beneficial physical activity is 150-200 minutes of 6-8 METs weekly, performed in 3-4 sessions. In the presence of cardiovascular disease, physical exercise programs are the basic elements of rehabilitation. Competitive and exhausting sport, or some recreational activity (> 20 hours of 15-20 METs) may produce electrical and functional changes that are morphologically very similar to the right ventricular arrhythmogenic cardiomyopathy (ARVC) and may cause sudden cardiac death. Emotional stress and Type-D personality may also cause sympathetic nervous overflow. Excessive sport performance, like Marathon run or triathlon may also be a challenge between sympathetic and parasympathetic pathologic nervous interactions with very rare, but fatal cardiovascular complications.

Keywords: Regular physical exercise, competitive sport, Marathon run and triathlon, sympathetic – parasympathetic nervous interaction, sudden cardiac death.

The most characteristic effect of the psychical exercise manifested on the cardiovascular regulation is the augmentation of cardiac output in proportion with the metabolic demand. The basic principle of the regulation that the core temperature of the human body stays constant (hyperthermia cannot occur) and the blood flow and oxygen supply of life important organs are within the normal limits during exercise. The increase in cardiac output and pulse rate as well as the augmentation of venous return - taking also into account the increased pulse volume in trained individuals - up to reaching the steady state (plateau phase), is close to linear. The

number of sympathetic neuron discharges are also increasing, the plasma concentration of epinephrine and norepinephrine are augmenting.

The homodynamic response is dependent on the intensity of the stress and the measure of muscular contraction as well. The maximal augmentation of cardiac output in untrained individuals is about fourfold (20-22 L/min), in well trained subjects it can be eightfold and the cardiac output could reach the 35-40 L/min value (Rowel, 1993).

The beneficial effects of physical exercise can be divided into early (developed within weeks, months), or late (manifesting in month, years) effects.



Early effects:

- a. *Endothelial effects:* Increased vasodilator effects of small arterioles and arteries with an increase of nitric-oxide (NO) production (Hambrecht et al., 2003).
- b. *Increased parasympathetic influence:* The increase in heart rate variability in a sensitive indicator of elevated parasympathetic (nervus vagus) tone. As a well known effect of training programs the heart rate variability is increased – as well as in healthy subjects, and patients suffering in cardiovascular diseases –, and the incidence of sudden cardiac death is decreased.
- c. *Coagulation factors:* High intensity cardiovascular exercise augments the concentration of coagulation factors, mostly factor VIII. The effect can be observed after attaining the 95-100 per cent of the maximal oxygen uptake and can be observed at least during one hour. In the case of more sustained stress a surplus production of fibrin and thrombin and an increase in platelet aggregation can also be observed.

Late beneficial effects:

- a. *Effects on lipoproteins:* according to a metaanalysis, due to physical exercise the concentration in total cholesterol decreased by 6.3 per cent, low-density cholesterol (LDL) diminished by 13.4 per cent; the protective high-density cholesterol (HDL) increased by 5.0 per cent. Training program decreases LDL cholesterol and increases in HDL cholesterol subfraction.
- b. *Caused by aerobic exercise:* the systolic and diastolic blood pressure decrease. A 2 per cent decrease in diastolic pressure mitigates by 6 per

cent the occurrence of cardiac, and by 15 per cent the incidence of cerebral death caused by stroke.

- c. *Diabetes mellitus:* Physical exercise has a multifactorial beneficial effect on the stabilization of glucose metabolism; the hepatic glucose production is decreased, the insulin resistance is mitigated. The glucose consumption of the skeletal muscle is increasing, the capillary density is augmenting and on physical stress the capillary flow became stronger and more effective.
- d. *Obesity:* Regular physical activity decreases obesity and prevents overweight. Regular physical exercise combined by decreased caloric intake effects in a larger and more stable body weight decrease (Malik et al., 2004).
- e. *Metabolic syndrome and chronic inflammation:* Metabolic syndrome (hypertension, overweight, hyperlipidaemia, insulin resistance is a very important disease entity, first of all, because it is linked to the development of ischemic heart diseases (acute coronary syndromes) and also because of its higher incidence than the sum of their risk factors. In its prevention, the regular physical exercise decrease incidence and measure all of its predisposing factors and if the training program and effective weight loss (more than 10 per cent of the body weight) can be attained the metabolic syndrome may cease.

Regular physical exercise or training program (optimally, at least 4 times 40 -60 minutes submaximal /70%/ of the number 220, minus age number) dynamic exercise combined by light stretching, 10-20 minutes, two times weekly) Its beneficial physiologic and pathophysiologic effects are summarized in *Table 1*.

Table 1: Physiologic effects off the regular exercise

Skeletal muscle	Capillary density is increased Oxydative enymes are increased Myoglobine concentration is increased Number and size of mitochondria are increased
Heart muscle	Oxygen supply increased (ECG, perfusion) Resting pulse rate and blood pressure are decreased Working capacity is increased
General	Body weight decrease Glucose tolerance is increasing HDL cholesterol is increasing

Vascular biologic effects of the training programs

Regular physical exercise increases coronary flow. This beneficial effect is caused by structural (remodeling) and functional modulations; the tissue perfusion of the heart muscle is augmented, the progression of the coronary atherosclerosis process is slowed or even stopped, the pulse volume and cardiac output is augmented. The qualitative and quantitative measures of the diminution of the coronary artery plaques and the demodulation achieved, can be visualized by coronary CT and MRI investigations.

The desirable weekly measure of physical activity (150-200 minutes in the form of 6-8 metabolic equivalent (MET), performed in 3-4 sessions) does not mean an excessive effort (for example in the form of quick walking, or jogging (4.6 -8.0 km/hour speed), or biking (by 15-20 km/hour) (Sharma et al., 2015). In a more advanced age, if the physical activity is significantly decreased, even a lesser amount of workload (about 4 MET) is sufficient to decrease the expected mortality rate by 12-20 per cent.

In the presence of cardiovascular disease the physical exercise programs are the basic elements of the rehabilitation. Beside the cardiovascular system, the regular physical exercises and training programs are also diminishing the incidence of the prostatic and female breast cancer (Sharma et al., 2015), and they have a role in the prevention of the aggravation of dementia and osteoporosis. Patients, who are participated in the training programs detailed above, have a 3-year expected lifetime prolongation, and that is why the regular physical exercise could be regarded the cheapest and most effective health-preserving and life-prolonging method.

Competitive (and some recreational) athletes perform a way above the current European and American guidelines and many of them engage in over 20 h of intense exercise (15 MET) per week. Such intense exercise requires a sustained – 5-6 fold – increase in cardiac output. It produces a unique electrical and functional cardiac adaptation that is termed the „athlete’s heart”, but in some cases this demodulation could be very similar to the genetically defined right ventricular arrhythmogenic cardiomyopathy (ARVC). During such a sustained left and right ventricular overload, the right ventricular peak pressure values could also

be higher than the left ventricular ones (La Gerche et al., 2015) and they can produce life-threatening ventricular arrhythmias (Zaidi & Sharma, 2015) causing right ventricular failure, circulatory collapse and terminal signs. La Gerche et al (2015) applied stress magnetic resonance imaging (MRI) method to visualize the right and left ventricular performance during peak exercise (Zaidi & Sharma, 2015), and they found, that even in these cases of maintained right ventricular ejection fraction (EF) the diminished right ventricular reserve causes the right ventricular arrhythmic risk, and primarily the declining right ventricular contractile decline may cause the life threatening arrhythmias and sudden cardiac death (SCD). Their final conclusion, that during sustained maximal physical stress, right ventricular failure may occur, that may conclude in fatal right ventricular arrhythmias and SCD. The study has also pointed out, that among 17 elite sportsmen suffered in right ventricular failure manifested on peak exercise, 8 had to receive implantable cardiac defibrillator (ICD) – and by that they could continue their sport activity. According to our present knowledge, the cases of genetically determined ARVC and right ventricular arrhythmogenic cardiomyopathy acquired by extreme sport activity cannot be differentiated even by exercise echocardiographic or exercise MRI methods. Recently, Szelid et al (2015) pointed out in Hungarian sportsmen, that the polymorphism of nitrogen monoxide (NO), the NOS3 Glu298Asp may also have a causative role in the development of the sport- induced ARVC.

The association of emotional stress and diseased heart condition and cardiac death is a longstanding human observation. Classical and from medical standpoint also well-analyzed event was the extremely strong earthquake that occurred on 17 January 1994 struck the Los Angeles area. The earthquake itself lasted for 10-20 s, and as a direct consequence. 57 people lost their lives, and during the same day, 24 sudden cardiac deaths were recorded, the rate more than five times higher than the usually expected (Stämpfli & Enseleit, 2015). Similar findings, that are attributed to an experience of extreme stress, sympathetic surcharge and catecholamine load leading to SCD are triggering by missile attacks, or less severe conditions such as anger or watching a football game. Even in 1915 Walter Cannon, in

the description of animal “fight or flight” reaction explained, that whenever a creature is faced with a potentially harmful or life threatening situation several adaptations occur, enabling the animal to react to this, with better chances of success. Later on, *Hans Selye* (1955) was the first to use the term in a biological context. According to his explanation, a certain “stressor” may be tolerated by one creature, whereas others are not able to cope with it. Selye investigated the influence of different stressors and to the reproducible sequence of the response to the stress he applied the terms “alarm”, “resistance”, and “exhaustion”. All of them are very comparable to the behavior and reactions that can be observed in competitive sport, and at least partly – may help to explain unexpected changes in performance, or even SCD. This explanation is also strengthened by the data of the timing of syncope events during Marathon run, which occurs mostly during the terminal 30 min of the run, and moreover during first 30 mins after having passed the Marathon line (*O'Connor et al., 1999; Maron et al., 2009*). This timing of tragic event may reflect to the interaction of sympathetic and parasympathetic interaction in the cardiovascular

system (sudden bradycardia, prolongation and dispersion of depolarization in the ventricular tissue and catecholamine overload as a trigger) as a causative factor. Similar pathomechanism can be suspected during the unexpected SCD described during in triathlon competitions, promptly after the start (jump into the cold water). The possible background of this fatal event can be the follows: Extreme catecholamine load before the start (tachycardia), diving into the cool water that triggers trigeminal-cardiac (diving) reflex, it causes bradycardia, prolonged depolarization. Finally interaction of reflex provoked bradycardia, inhomogenous depolarization and catecholamine load, which trigger life threatening ventricular tachycardia (VT) – which may cause SCD (*Harris et al., 2010; Williams et al., 2015*).

It is important to emphasize, that the occurrence of SCD is at least 3-6 fold more frequent in hobby sport activity compared to fatal events occurred in competitive sport. It is also proved that in competitive sport the main cause of the unexpected is mostly related to the individual cardiovascular risk status.

Mental stress and heart diseases (Proietti et al. Future Cardiol 2011;7:425-37)

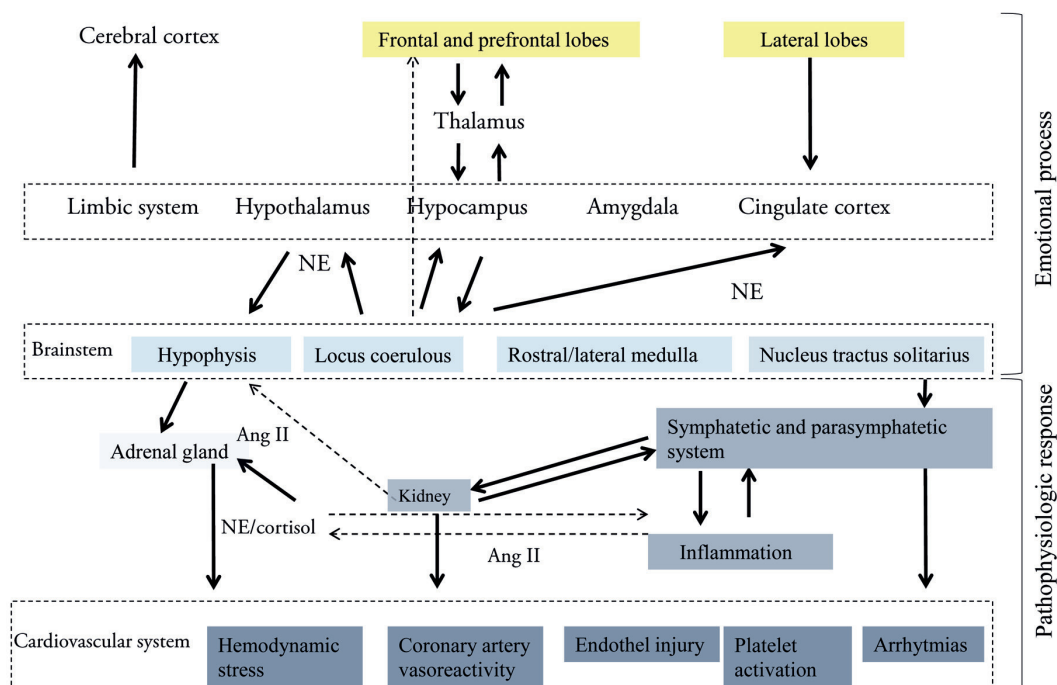


Figure 1: Pathophysiologic interrelationship of mental stress and cardiovascular diseases.
(After *Pioretti et al: Future Cardiol 2011;7:424-37*)

Several lines of research support the biological plausibility of Type D (distressed) personality as a risk marker. Type D refers to a general propensity to stress, characterized by negative emotions, and social inhibition, and more frequent cardiac death. The European and American guidelines are listed Type D patients as candidates for metabolic dysfunction, with increased severity of coronary plaques, increased risk for cardiac arrhythmias and Tako-Tsubo cardiomyopathy (Denollet et al., 1996; Denollet & Kupper, 2015). Since they are much more prone to mental stress and cardiovascular diseases their screening among the candidates for racing and hobby (health-enhancing) sport activity would be effective.

Hypertension as the most important risk factor for cardiovascular diseases has a 40-45 per cent general incidence in the European population. One of its most important organ manifestation the heart disease in general, and more specifically the left ventricular hypertrophy (LVH), which is an actual blood-pressure independent predictor for the SCD (Messerli, 1999). Presence of LVH and high blood pressure (presence of LVH and high blood pressure RR > 140/90Hgmm) is also a predisposing factor for complex ventricular arrhythmias. Appropriate drug / or nonpharmacologic treatment (regular physical exercise, body weight reduction) of hypertension is also attenuate risk for arrhythmias associated with LVH. Ventricular ectopic beats are strong predictor for SCD; that is why in the screening of participants of hobby and competitive sport the pharmacological and non-pharmacological prevention of ventricular arrhythmias is of primary importance. In the presence of ventricular arrhythmias active sport activity cannot be permitted.

References

- Rowel, L. B. (1993). *Human Cardiovascular Control*. New York, Oxford University Press.
- Hambrecht, R. et al (2003). Regular physical activity improves endothelial function in patients with coronary artery disease. *Circulation* 107, 3152-3158. <http://dx.doi.org/10.1161/01.CIR.0000074229.93804.5C>
- Malik, S. et al (2004). Impact of metabolic syndrome on mortality from coronary heart disease. *Circulation* 110, 1245-1250. <http://dx.doi.org/10.1161/01.CIR.0000140677.20606.0E>
- Sharma S, Merghani, A., Mont, L. (2015). Exercise and the heart: the good, the bad, and the ugly. *Eur Heart J* 36;1445-1453. <http://dx.doi.org/10.1093/eurheartj/ehv090>
- La Gerche, A. et al. (2015). Exercise-induced right ventricular dysfunction is associated with ventricular arrhythmias in endurance athletes. *Eur Heart J* 36, 1998-2010. <http://dx.doi.org/10.1093/eurheartj/ehv202>
- Zaidi, A., Sharma, S. (2015). Arrhythmogenic right ventricular remodelling in endurance athletes: Pandora Bocs or Achilles wheel? *Eur Heart J* 36, 1955-1957. <http://dx.doi.org/10.1093/eurheartj/ehv199>
- Szelid, Zs. et al. (2015). Right ventricular adaptation is associated with the Glu298Asp variant of NOS3 gene in elite athletes. *Plos One* Oct 30;10(10):20141680. <http://dx.doi.org/10.1371/journal.pone.0141680>
- Stämpfli, S.F., Enseleit, F. (2015). Stress and cardiovascular mortality; An evident, but underestimated connection. *Eur Heart J* 36(28), 1781-1782
- Cannon, W.B. (1936). The role of emotions in disease. *Ann Int Med* 9, 1453-1465.
- Selye, H. (1955). Stress and disease. *Science* 122, 625-631.
- O'Connor, F. G. et al (1999). Exercise related syncope in the young athlete: reassurance, restriction or referral? *Amer Fam Physician* 60, 2001-2008.
- Maron, J. et al. (2009). Sudden death in young competitive athletes. Analysis of 1866 death in the United States, 1980-2006. *Circulation* 119, 1085-1092. <http://dx.doi.org/10.1161/CIRCULATIONAHA.108.804617>
- Harris, K. M. et al. (2010). Sudden death during triathlon. *JAMA* 303, 1255-1257. <http://dx.doi.org/10.1001/jama.2010.368>
- Williams, T. M. et al. (2015). Exercise and death alters bradycardia and incidence of cardiac anomalies in deep-diving murin mammals. *Nature communications* No6055 <http://dx.doi.org/10.1038/ncomms7055>
- Denollet, J. et al. (1996). Personality as an independent predictor of long-term mortality in patients with coronary heart disease. *Lancet* 347, 417-421.
- Denollet, J., Kupper, N. (2015). Stress and the heart: the role of type D personality in personalized care. *Eur Heart J* 36:1783-1786.
- Messerli, F.H. (1999). Hypertension and sudden cardiac death. *Am J Hypertens* 12, 181S-188S.